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Commotio Cordis: Prevention and Survival

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Commotio Cordis: Prevention and Survival

A Senior Honors Thesis

Submitted in Partial Fulfillment of the Requirements
for Graduation in the Honors College

By
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The College at Brockport
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Introduction

Commotio cordis (CC) is a potentially fatal cardiac event that occurs after a blow to the chest. It is most commonly seen in youth sports where projectiles are approaching an athlete such as baseball, lacrosse, hockey, softball, soccer, and karate.¹⁻⁴ Commotio cordis occurs in healthy individuals with no prior history of cardiac conditions, adding to the unpredictability of CC. Commotio cordis is the second most common cause of sudden death in young athletes, while hypertrophic cardiomyopathy is the first.⁵⁻⁷ Due to the “perfect storm” of events that are necessary for CC to occur, it is a relatively rare. However, CC still accounts for twenty percent of sudden death cases in youth.⁷ While CC is a relatively rare occurrence, rates of CC incidences have been increasing over the last 20 years.⁷ However, this does not necessarily mean that cases of CC are becoming more common than in previous years. Since this data is only based off of recorded incidences, many believe this increase is due to increased recognition of CC.⁷ With more cases of sudden death being recognized as CC, more incidences are being reported.

It has been shown that younger athletes are more susceptible to CC due to their pliable chest walls.⁷⁻¹⁰ The average age of CC victims in reported incidents is approximately 16 years old.⁷ The first description of CC can be traced back to 1763, while the first evaluation of CC did not occur until 1932.⁷ The time lapse from

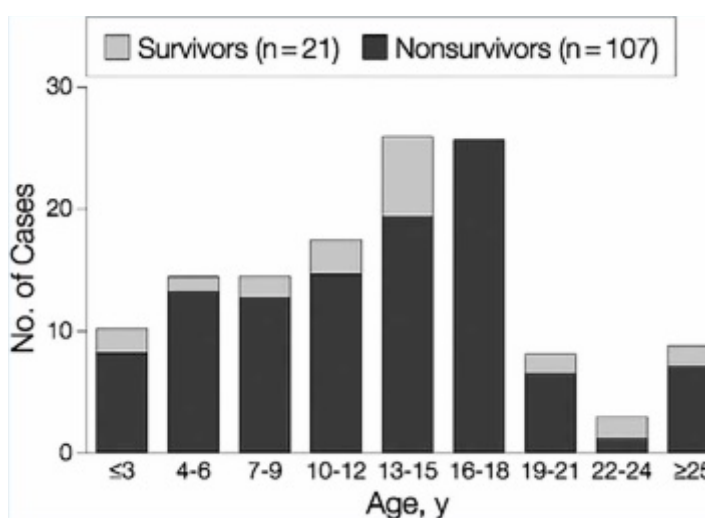


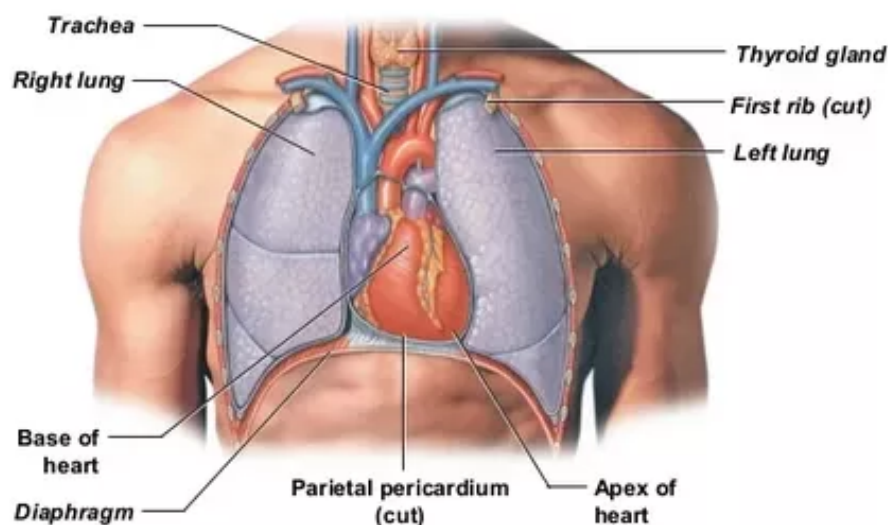
Figure 1. Age and rates of survival of reported commotio cordis incidences.¹

description to evaluation may be the result of the perplexing nature of this cardiac event, leading

many cases to go unrecognized before research was possible. While CC is a relatively rare occurrence, rates of CC incidences have been increasing over the last 20 years; many believe this is due to increased recognition and reporting.⁷

Anatomy

The heart is the main organ of the circulatory system. It is roughly the size of a clenched fist and is located behind the sternum, near the anterior chest wall.¹¹ The heart is anatomically protected by the sternum, ribs, and surrounding musculature. Since the majority of the pectoralis major originates on the lateral sternum, it can be said that the pectoralis major is one of the main



muscles that can act to protect the heart from impacts. The intercostal muscles may also have a role in anatomical protection.

The heart sits within the cardiac notch of the left lung, between

Figure 2. The heart's location within the thoracic cavity.²

the two pleural cavities. It is surrounded by the pericardium, which acts as a balloon to stabilize the heart in its proper position.¹¹ The pericardial cavity is full of pericardial fluid, which reduces friction between the heart and the surrounding structures when the heart beats.¹¹ Inside the pericardium, the heart is covered by the outer epicardium, the myocardium, and the innermost endocardium. The myocardium is the muscular wall of the heart, containing cardiac muscle tissue, blood vessels, and nerves. This layer forms the atria and the ventricles of the heart.¹¹ The

heart is made up of four chambers; the right atrium, right ventricle, left atrium, and left ventricle. The atria are thinner than the ventricles and have the ability to expand to great amounts.¹¹

The left atrium collects oxygenated blood returning from the lungs, through the pulmonary vein, and

pumps it through the

mitral valve into the left

ventricle. The left

ventricle is then

responsible for

pumping this

oxygenated blood to the

rest of the body, via the

aorta, first passing

through the aortic

valve. The right atrium

is responsible for

collecting deoxygenated

blood from the body, through the vena cava, and pumping it through the tricuspid valve into the

right ventricle. The right ventricle must then pump the deoxygenated blood to the lungs, via the

pulmonary artery, first passing through the pulmonary valve. Once inside the lungs, the blood

can become oxygenated once more. Cardiac muscle requires oxygenated blood just as much as

any muscle in the body does. Cardiac veins and arteries form a circulation system to supply

blood to the muscle tissue of the heart. As the level of exertion during activity increases, so too

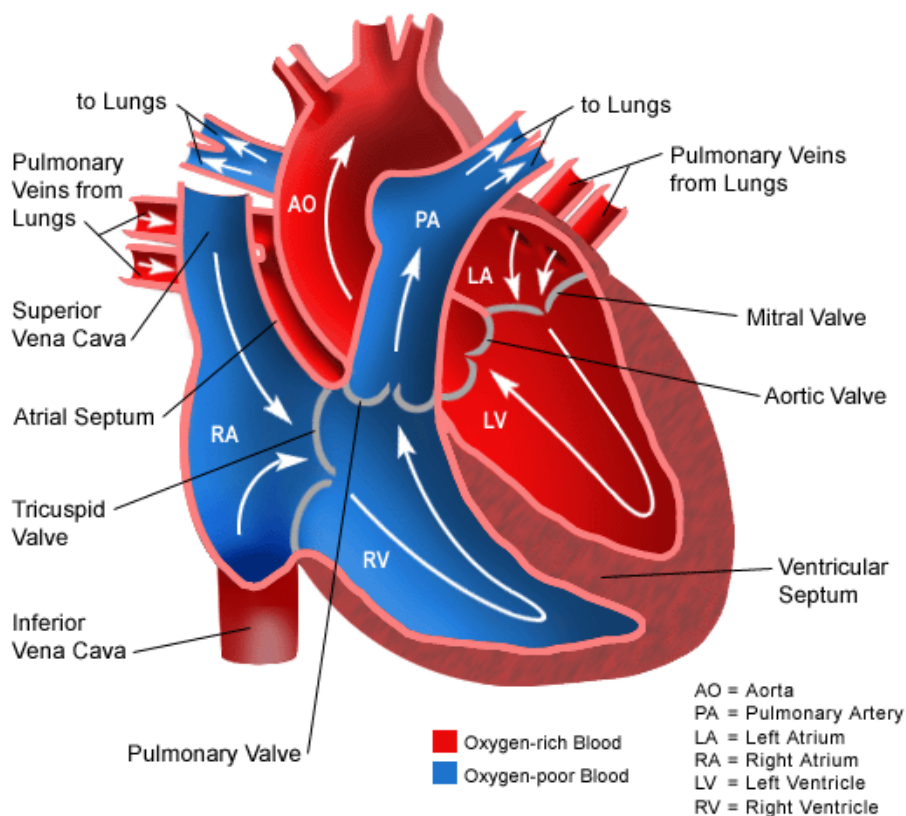


Figure 3. The flow of blood through the heart.³

does the heart's demand for blood. According to Martini et al., the blood flow to the heart's musculature during maximum exertion can increase to nine times the resting amount.¹¹

Physiology

A heartbeat, otherwise known as a cardiac contraction, is the contraction of the entire heart, beginning with the atria, followed by the ventricles in series.¹¹ In order for the heart to beat automatically, there must be a conduction system that sends electrical impulses to the chambers so they can contract without needing direct neural stimulation. This conduction system consists of the sinoatrial (SA) node, the atrioventricular (AV) node, and conduction cells. The

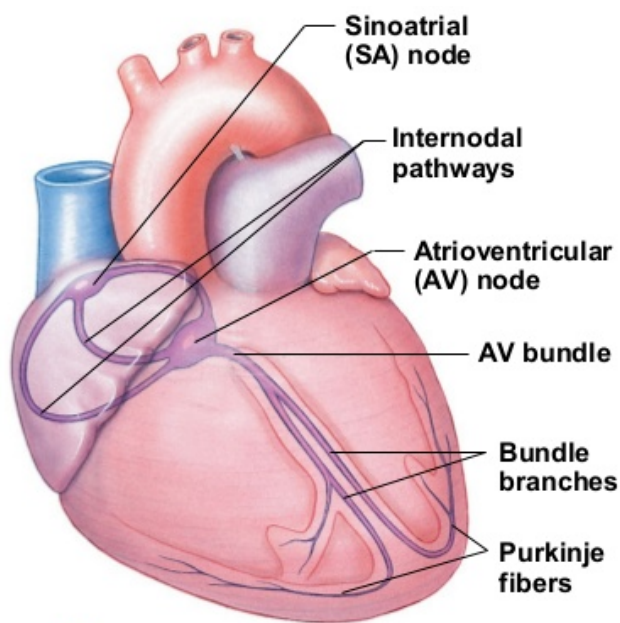


Figure 4. The components of the cardiac conduction system.⁴

SA node is located in the right atrium and acts as a pacemaker by generating action potentials and sending them through the atria.¹¹ From the atria the impulse spreads and reaches the AV node in the floor of the right atrium. There is a 100-millisecond delay once the impulse reaches the AV node before it is conducted via bundle branches to the Purkinje fibers and distributed to the ventricles.¹¹ This delay is important because it coordinates

the atria to contract followed by the ventricles in series instead of at the same time.¹¹ This allows the blood to adequately move from the atria into the ventricles and get pumped out of the heart. These electrical impulses are generated at regular intervals so the heart can continuously pump

throughout life. The period from the start of one heartbeat to the start of the next is called the cardiac cycle.¹¹

The electrical charges that occur during the cardiac cycle are great enough to be measured by electrodes on the skin's surface with an electrocardiogram, or ECG.¹¹ An ECG features the P wave, QRS complex, and the T wave. The P wave occurs first and signifies the depolarization of the atria. Once the atria are depolarized they contract, pumping blood into the ventricles.¹¹ The QRS complex appears next of the ECG, measuring the depolarization of the ventricles. The amplitude of the QRS complex is greater than that of the P wave because the ventricles are much bigger than the atria, requiring a larger stimulus. The ventricles begin contracting following the peak of the R wave.¹¹ The T wave follows the QRS complex, measuring the repolarization of the ventricles. The repolarization of the atria is not seen in an ECG because it occurs during the same time as the QRS complex and therefore is not picked up or recognized by the leads.

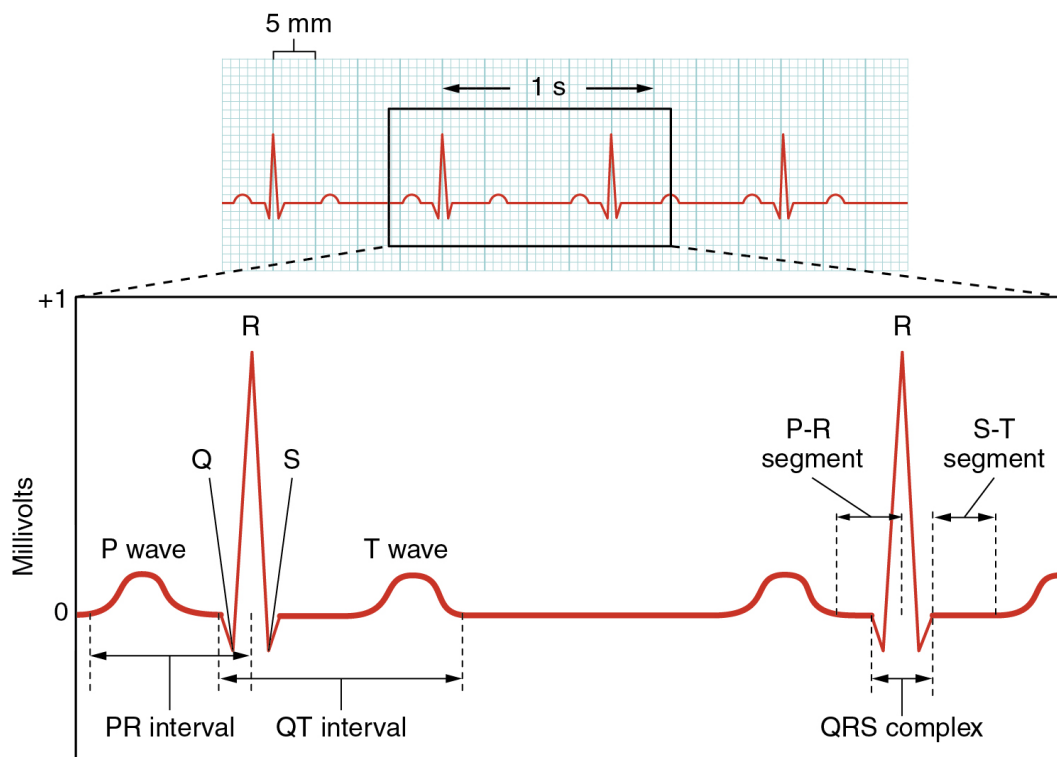


Figure 5. An electrocardiogram of a normal sinus cardiac rhythm.⁵

Etiology

Commotio cordis is caused by a blunt, non-penetrating trauma to the chest wall, over the heart.^{3, 8, 10, 12} The center of the left ventricle is the most vulnerable to impacts that lead to CC.⁷

Trauma to the precordial area must occur during an electrically vulnerable period, 10-30 milliseconds before the peak of the T wave, in order to be considered CC.⁵⁻⁷ Following impact, there is an absence of structural damage to the heart, ribs, and sternum, due to the relatively low

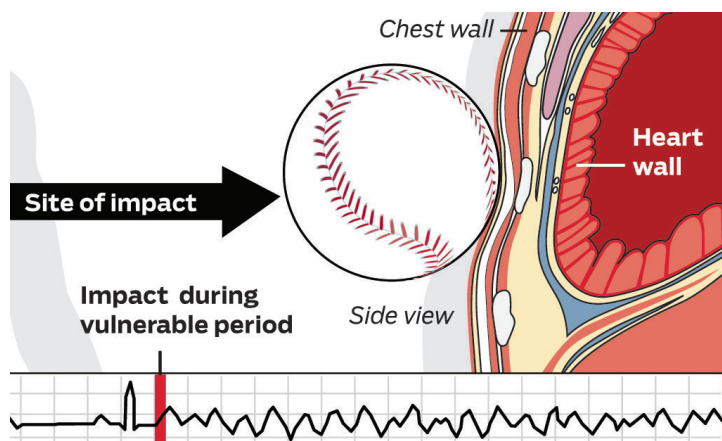


Figure 6. Impact of a projectile over the precordial area, initiating commotio cordis.⁶

speeds of impact.^{2, 12} Commotio cordis inducing blows are most commonly the result of dense projectiles moving at 30-50 MPH.^{2, 4, 5, 7, 12} There is a direct correlation to the density of the projectile and the risk of CC, with denser projectiles leading to higher rates of death.^{5, 6, 9}

Commotio cordis occurs in healthy individuals with no prior history of cardiac conditions.^{3, 8-10} The young population is most vulnerable to CC due to their pliable chest walls, prior to full development, allowing more energy to be transmitted from the projectile directly to the heart.⁸⁻¹⁰ As a result of this, CC is the second most common cause of sudden death among young athletes.^{5, 6} Little league baseball unfortunately combines young athletes and their pliable chest walls with pitches approaching the plate at speeds of 30-50 MPH. Due to the nature of the sport and the age of the athletes, little league baseball has the highest rate of incidence of CC.^{7, 13}

Competitive and recreational sports are the most common activities that give rise to CC. Commotio cordis incidences have been reported during baseball, softball, hockey, football, soccer, lacrosse, and cricket.^{1-4, 7} While the ball used in cricket is very similar to a baseball, the rate of incidence in cricket of CC is much lower than in baseball. This is believed to be the result of the different pitching techniques seen in the two sports.³ In baseball the ball is pitched from 45 feet to 66 feet and 6 inches, depending on the age of the players, with a strike zone ranging from the batter's chest and knee height. In cricket the ball is thrown from 22 yards (60 feet) and cross the batter below waist level.³ With the ball's lower trajectory, the chances of taking a ball to the precordial area are decreased in cricket.³

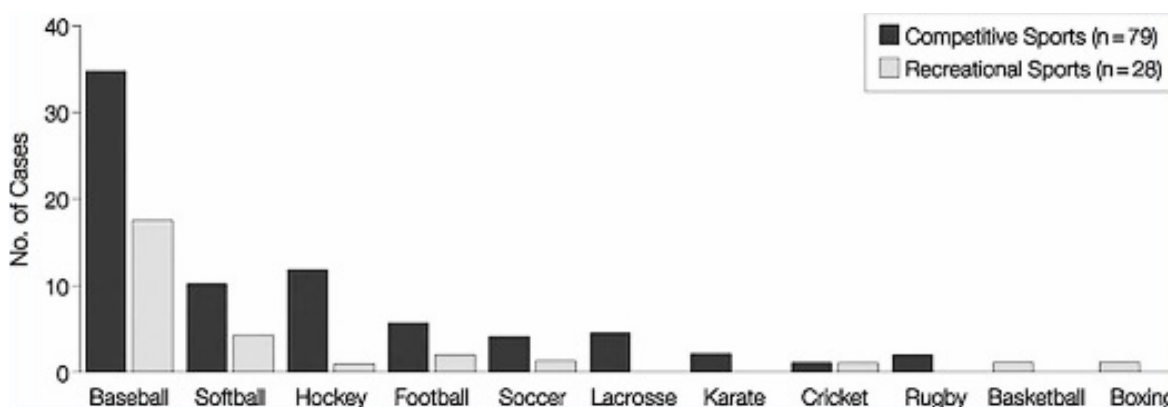


Figure 7. Sports during which a commotio cordis event has been reported.⁷

While most incidences of CC have been reported in sports, CC can occur from non-sport related events. Cases have also been seen related to child abuse and motor vehicle accidents.^{12, 13} In order for CC to occur, the location, timing, and force of impact, all have to combine to create the perfect storm of events. Commotio cordis can potentially occur during any activity if there is an impact is over the precordial area, 10-30 milliseconds before the peak of the T wave, by an object with great enough density and/or speed to create a great enough force. This unfortunately adds to the unpredictable nature of CC, not knowing when, how, and during what activity CC may occur.

Pathophysiology

Impacts to the precordial area from projectiles moving 30-50 MPH are not great enough to cause structural damage to the heart, ribs, or sternum. However, electrical interruptions occur, disrupting the cardiac cycle. Ten to thirty milliseconds before the peak of the T wave, the ventricles are repolarizing. It is at this point in time where the heart is most vulnerable to CC. Commotio cordis does not occur as the result of structural damages but instead is considered an “electrical phenomenon”.^{2,3}

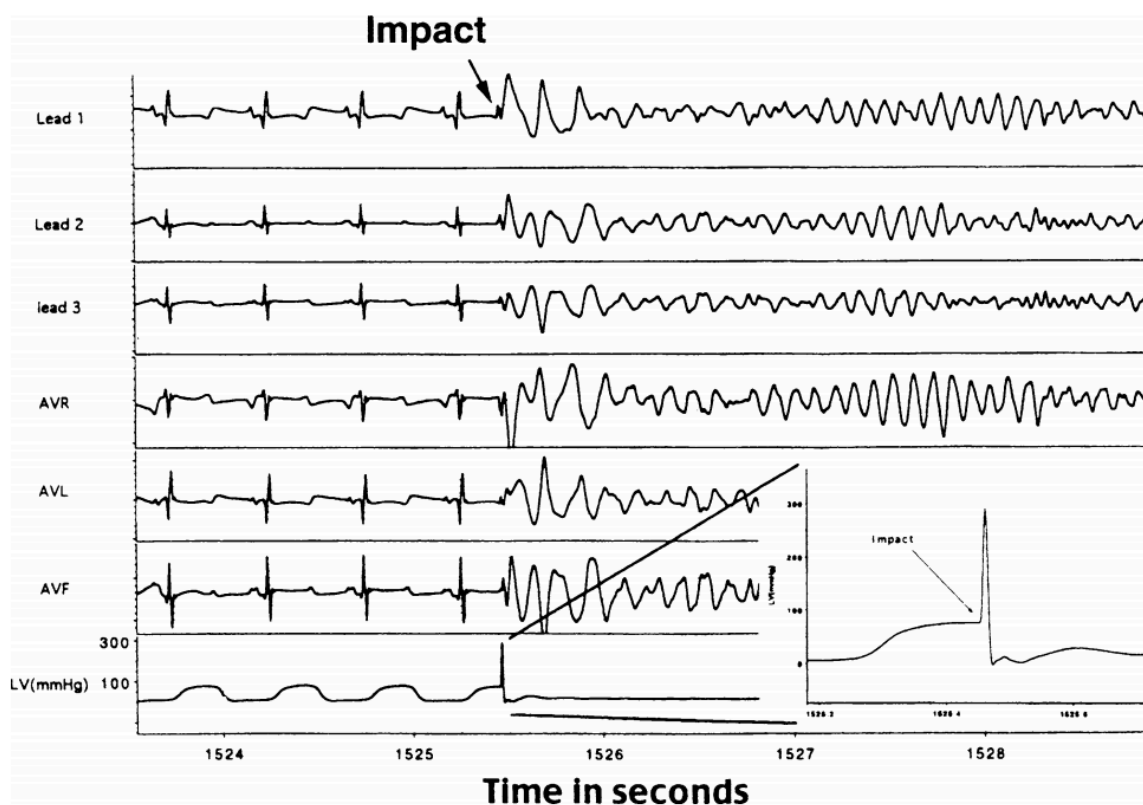


Figure 8. Ventricular fibrillation produced immediately on impact, when struck during the vulnerable time zone, 10-30 ms before the peak of the T wave.⁸

The force from the impact triggers an acute elevation of pressure within the heart's left ventricle.^{5,7} Mechano-sensitive ion channels within the heart are then triggered to open, sending an influx of ions into the cardiac cells.^{5,6} The influx of ions released interrupts ventricular repolarization, causing premature depolarization of the cardiac cells, which in turn triggers

ventricular fibrillation (VF).⁶ Ventricular fibrillation is a life threatening arrhythmia that is responsible for cardiac arrest.¹¹ It is the most common cardiac arrhythmia seen with CC.

Ventricular fibrillation has been described as a rapidly fatal useless quivering of the ventricles that prevents the heart from pumping blood.^{8, 11} Once the heart is in VF, the body will no longer get the oxygen it needs and brain damage can quickly occur.

It was suggested by Madias et al. that VF is induced when there is an electrical gradient great enough to activate myocardium that have an early local recovery of excitability, but fails to

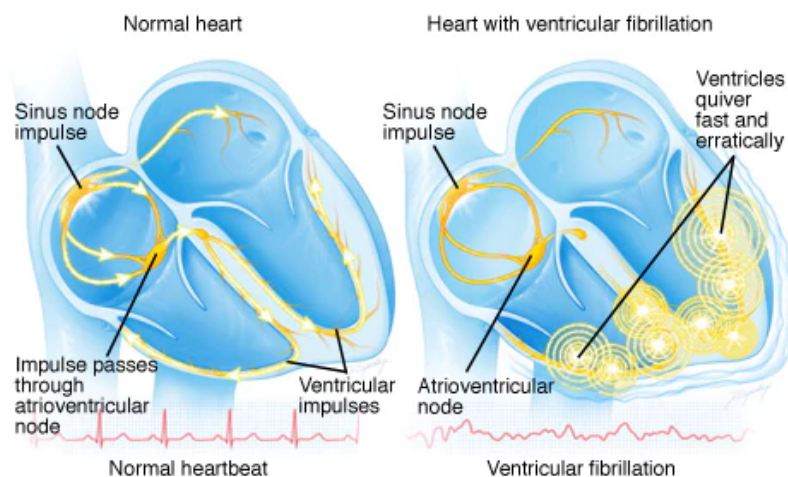


Figure 9. Rapid, erratic electrical impulses during ventricular fibrillation causing quivering of the ventricles.⁹

activate less-repolarized myocardium because of their local refractoriness.⁶ In other words, VF results from an unbalanced electrical stimulation that activates some myocardial cells but fails to activate other myocardial cells due to differences in refractory times,

caused by the influx of ions from the impact.

Upon impact the individual usually collapses immediately, but in some cases they may have a brief moment of lucidity prior to collapsing.^{3, 5} Once downed, the individual's condition deteriorates rapidly as the body is deprived of oxygenated blood. If defibrillation does not occur quickly, permanent damage to the brain and vital organs causing death, is inescapable. After death, upon postmortem examination, there is an absence of structural damage and

abnormalities. Superficial bruising over the point of impact has only been noted in a few cases.^{3, 6, 9}

Prognosis

Following impact, there is a short period of time before the individual reaches a point of irreversible damage. Death can occur within minutes if a trained individual does not administer proper aid.⁸ Surviving CC relies on cardiac defibrillation and cardiopulmonary resuscitation (CPR). Early application of chest compressions in CPR can sustain organ perfusion for a short period of time, until defibrillation is possible. Ultimately an automated external defibrillator (AED) is necessary to defibrillate the heart so it can return to sinus rhythm.⁵ According to the American Heart Association, the survival rate of an individual with CC declines by 10% for every minute that passes without defibrillation.^{2, 8} After 12 minutes, the average time it takes for emergency medical services to arrive, it is usually too late for revival.⁸

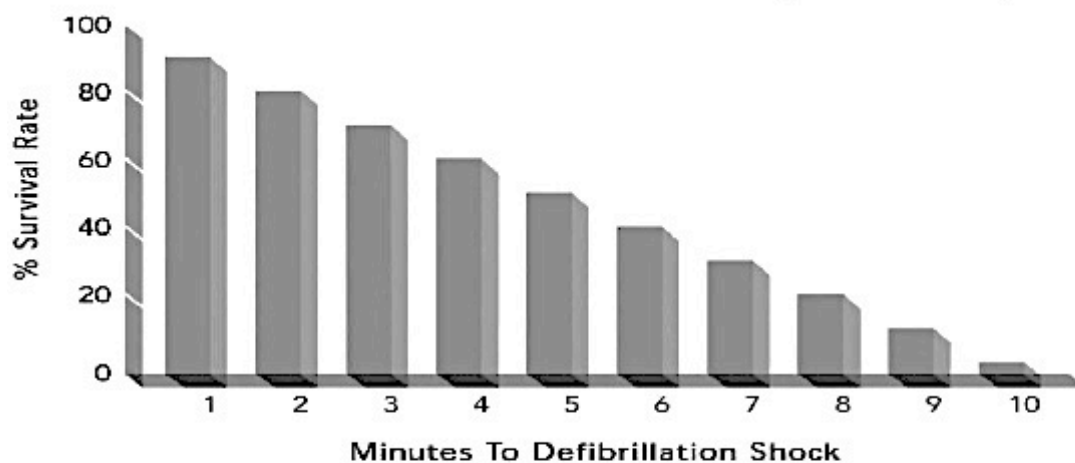


Figure 10. Time elapsed from impact to defibrillation and the survival rate associated.¹⁰

Individuals who survive immediate resuscitative efforts are not instantaneously out of danger. In the time following CC, there is still a chance that the heart's rhythm will revert back into arrhythmia. It has been proposed by Lateef that following a CC event; it is essential to the

survival of the individual that they have continuous ECG monitoring. This is due to the finding that directly following a CC incident, the most common cause of death, especially in the first 12 hours after the event, is dysrhythmia.³ While the individual has survived the immediate ventricular fibrillation from the impact of the projectile, their heart may acutely regress to erratic electrical impulses.

There is a case study by Berkson et al. of a 17-year-old male baseball catcher who was struck in the chest by a foul-tipped regulation baseball.⁵ According to the case study, following impact he stood up immediately, rubbed his chest, took nine steps, paused, and then collapsed onto his back.⁵ Resident physicians on site at the game attended to the victim immediately. The victim was unresponsive, with a weak carotid pulse. After 30 seconds he had no pulse, shallow infrequent respirations, and perioral cyanosis. The two resident physicians performed CPR while emergency services were activated. Nine cycles of CPR were performed, with no change noted in their reassessments. Four additional physicians arrived five minutes after the athlete's collapse, with an AED, which was immediately applied. One shock was given before resuming CPR. The athlete regained a strong pulse after approximately one minute and 75 additional chest compressions, but still remained unconscious and unresponsive with a left medial eye drift.⁵

Figure 11. Initial rhythm of ventricular fibrillation detected by the AED.¹¹

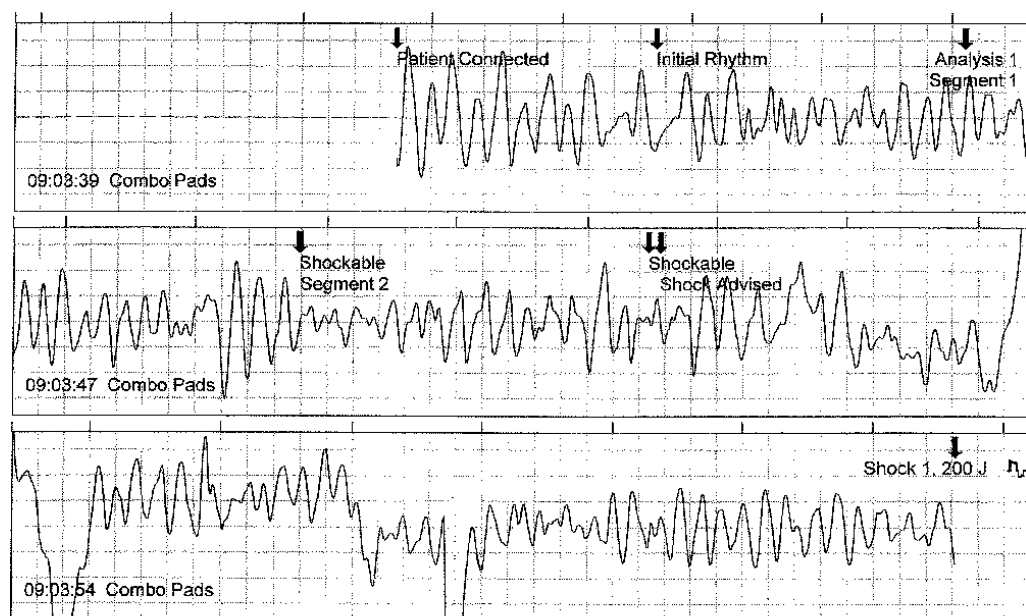


Figure 12.
Cardiac
rhythm after
advisable
shock was
delivered. ¹²

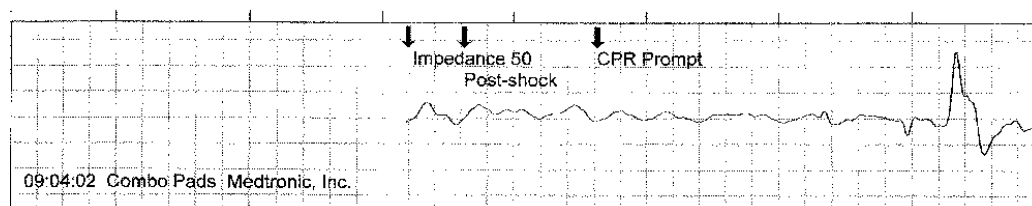


Figure 13.
Spontaneous
pulse is
recovered. ¹³



Nine minutes after the athlete's collapse, emergency medical services arrived and transported him to the hospital while the AED was taken for interrogation. The athlete seized once during transportation and once in the emergency room. He regained consciousness in the emergency room but remained confused and presented with vision loss, which later resolved. The athlete was admitted into the intensive care unit for observation. He presented with aphasia, altered memory, and delayed reaction time. His mental status returned to baseline within 48 hours, and he was symptom-free with the exception of mild chest soreness. The athlete was discharged and instructed to follow-up with a cardiologist after four to six weeks of no activity. ⁵

Survival rates of CC were extremely low in the past, prior to 1995. In 2002, according to the Commotio Cordis Registry, the survival rate had increased to 15% of reported cases. ^{2, 5, 6, 12}

Over the last 15 years, the CC survival rate has increased steadily. In 2012 the survival rate was greater than 50%. ⁴ This increase in survival is believed to be the result of increased recognition of CC. ⁸ When an event occurs and is quickly recognized as CC, the time elapsed between the trauma and intervention with CPR and defibrillation is decreased, which increases the chance of survival. ^A To further increase survival rates, the time elapsed from impact to defibrillation must continue to decrease. Factors to accomplish this include improved recognition of CC, increased availability of AEDs, and increased presence of individuals trained and willing to perform CPR and defibrillation using an AED. Without an improvement in these factors, CC survival rates may plateau, leaving athletes at risk for death.

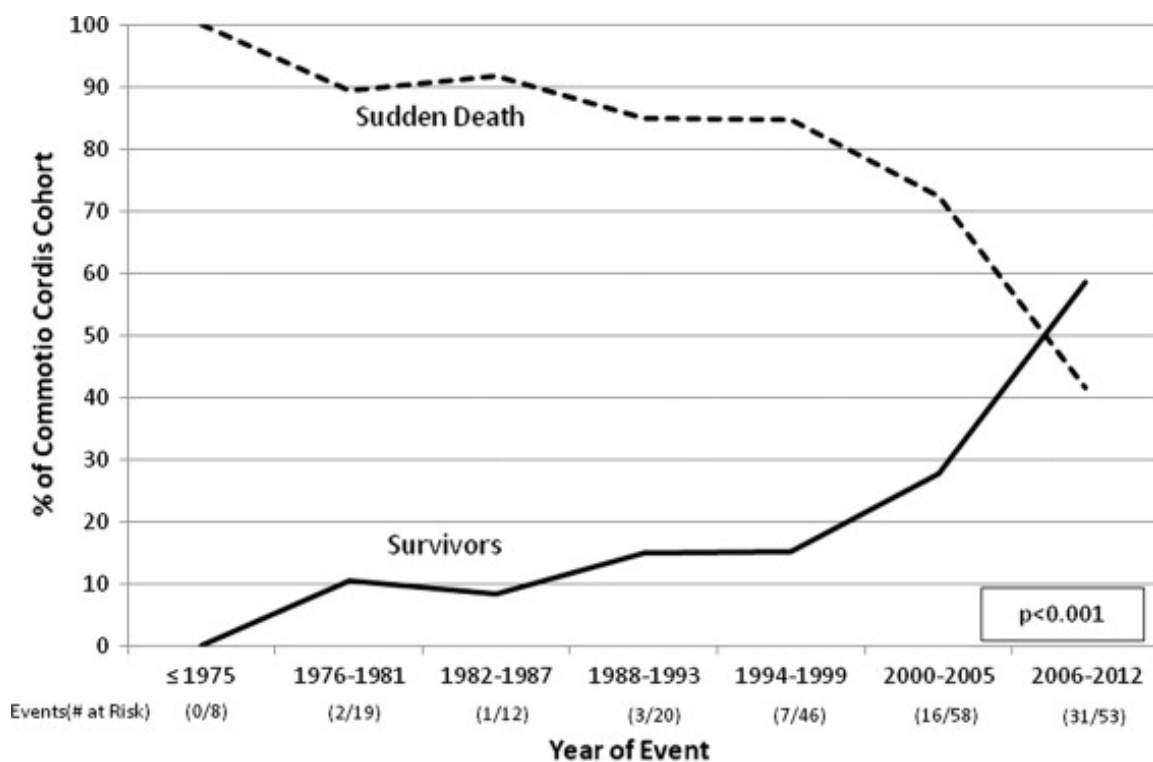


Figure 14. Increasing survival rates of commotio cordis according to the cases reported to the Commotio Cordis Registry. ¹⁴

Return to Play

An individual who survives CC should undergo a thorough cardiac evaluation to rule out any involvement of structural heart disease.^{4,7} This workup should include an ECG, exercise stress testing, echocardiogram, and magnetic resonance imaging.^{4,7} The individual may also undergo pharmacological testing for any genetic condition that results in an irregular heartbeat.⁴ If no underlying cardiac disease is present, an implantable cardioverter defibrillator (ICD) is not recommended.^{4,7} An ICD monitors heart rhythm; if it detects an arrhythmia it will send a shock to defibrillate the heart and restore sinus rhythm.¹⁴ Implantable cardiac defibrillators are typically used to prevent sudden death in patients with known conditions that put them at risk for ventricular arrhythmias.¹⁴ Commotio cordis is not a predictable condition and its reoccurrence is rare. Currently there is only one case of reoccurrence, making ICDs unnecessary in CC survivors.⁷

Return-to-play decisions should be made case-by-case, depending largely on the presence or absence of underlying cardiac conditions.^{4,7} If there is no cardiac condition present, the athlete may be able to return to sport. While the chances of reoccurrence are extremely small, some may advise not returning to sport until the chest wall has further developed and matured, in an effort to protect the individual.⁴ In regard to return-to-play decision involving contact sports, increased consideration of the chance of reoccurrence must be accounted for, due to the increased chance of taking another blow to the chest.⁷ It has been suggested by Kim that survivors of CC avoid caffeine, tobacco, alcohol, appetite suppressants, and any other substances that contribute to an irregular heartbeat.² In the case study mentioned previously, the athlete was cleared to return to play following a stress test, ECG, and echocardiogram all showing an

absence of abnormalities.⁵ The athlete had no adverse effects and went on to play Division II college baseball.⁵

Prevention

According to the U.S. Commotio Cordis Registry, 182 people have died from CC since 1995. Of these 182 incidents, 85 occurred during an organized sport and 32 of the victims were wearing a commercially available chest protector at the time of impact.^{1, 5, 7} It has been shown in many studies that commercially available chest protectors are ineffective at preventing ventricular fibrillation in CC.^{1, 5-8, 12} There are multiple theories to explain this failure, however two factors come up repetitively in the research, ineffective padding and improper fitting of chest protectors.⁵⁻⁸

In many of the reported incidences of CC where the athlete was wearing a chest protector, the impact occurred over a location that became unprotected when the chest protector became displaced as the athlete moved.^{5,6} This displacement occurs frequently when an athlete moves their hands above their head.⁷ The chest protector may also be improperly fitted due to the size of the child. Because children grow quickly, parents may opt into buying a chest protector that is slightly bigger than the recommended size for their child in an effort to save money and not have to buy a new chest protector the following year when they are taller and bigger. In theory this may sound like a flawless idea, bigger chest protector, more protection, but this is not the case. When a chest protector does not fit properly, there is a potential for the chest protector to not cover the full chest and leave areas vulnerable to getting hit. When a chest protector is too big for an athlete, it is very likely not tight enough and can displace easily when the athlete moves, especially when raising the hands above the head, because it is so loose. In order for proper fitting of a chest protector, athletes should try on the equipment before they

purchase it, and follow the manufacturers guidelines.⁸ Chest protectors that are designed for one sport are not interchangeable for other sports unless specifically stated in the manufacturers guidelines.

If the chest protector fits properly, it can still fail to protect the athlete from CC due to ineffective padding. A study completed by Weinstock et al., prior to 2006, tested the effectiveness of commercially available baseball and lacrosse chest protectors for prevention of CC.¹⁵ Weinstock et al. used 12 different chest protectors on animal models, with impacts occurring during the vulnerable period of repolarization, at 40 MPH. Baseball chest protectors were tested with regulation baseballs as the projectile, and lacrosse chest protectors used regulation lacrosse balls. It was found that none of the chest protectors commercially available showed a significant ability to decrease ventricular fibrillation when compared to controls with no chest protector.¹⁵ Weinstock et al. suggested that the materials and design of the chest protectors must improve in order to reduce the occurrence of ventricular fibrillation.¹⁵

Similar data was also found by Madias et al. in a study where portions of 12 baseball and lacrosse chest protectors commercially available were tested using impacts of 40 MPH on swine models.⁶ None of the chest protectors significantly decreased the occurrence of ventricular fibrillation. It was also found that the pressure in the left ventricle produced by the impact was linearly correlated to the probability of ventricular fibrillation.⁶ Due to these findings, Madias et al. concluded that the ineffectiveness of the designs is likely caused by the inability to reduce peak left ventricular pressure with impact.⁶

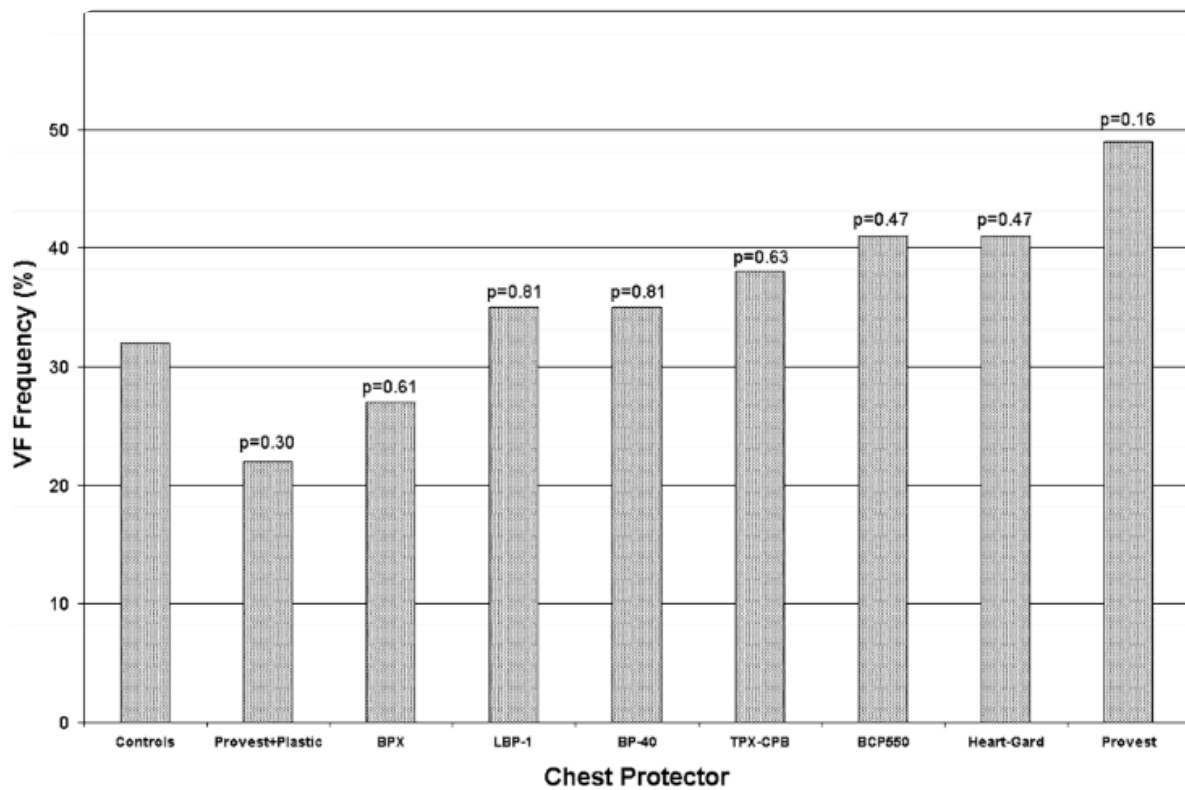


Figure 15. Incidence of ventricular fibrillation with baseball chest protectors vs. controls.¹⁵

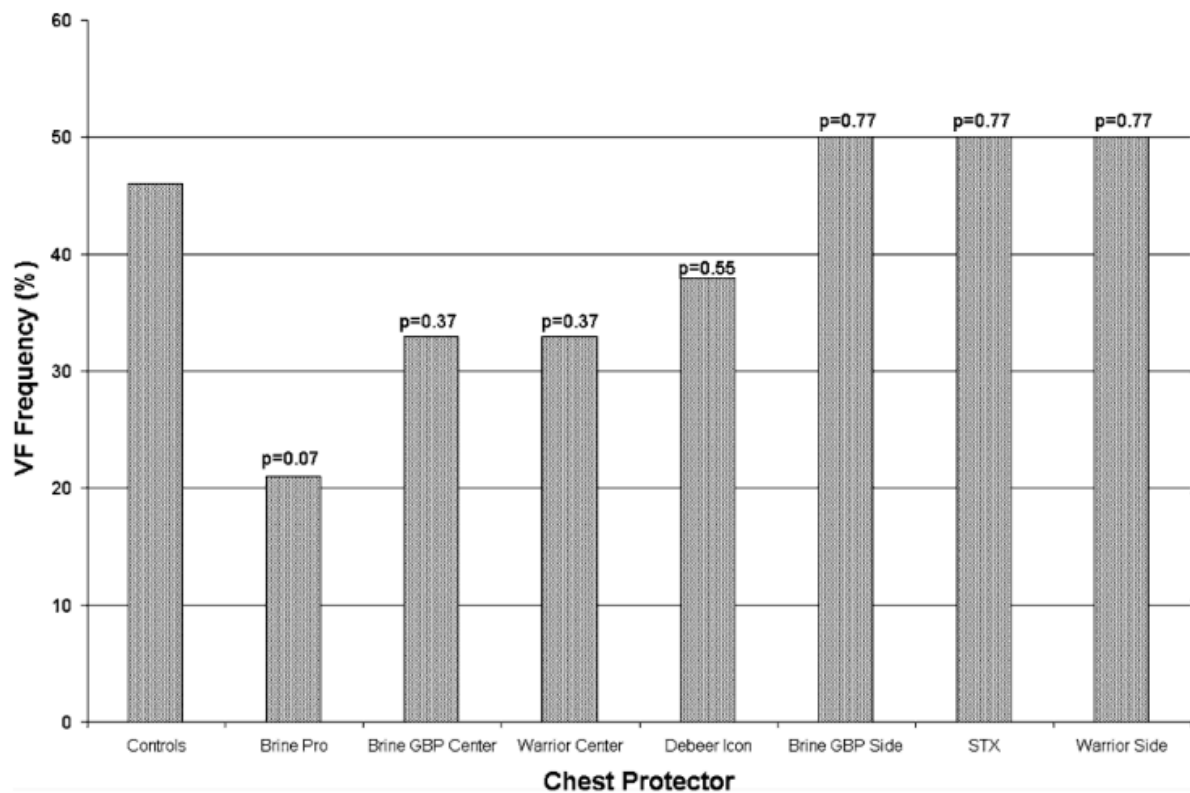


Figure 16. Incidence of ventricular fibrillation with lacrosse chest protectors vs. controls.¹⁶

The chest protectors tested in both of these studies were composed of very similar materials. At the time, many if not all commercially available chest protectors consisted of a layer of closed-cell or open-cell foam, covered by fabric or a hard shell.^{1, 3, 6} In an effort to better understand the mechanical properties causing these chest protectors to fail, Drewniak et al. analyzed the data collected from Weinstock et al. One chest protector showed a trend toward reduced occurrence of ventricular fibrillation, and it was composed of slightly different material than the others. This led Drewniak et al. to the idea that perhaps there is a mechanical property of the chest protectors that can be correlated with decreased occurrence of ventricular fibrillation.¹² Through the study it was discovered that there was a significant decrease in occurrence of ventricular fibrillation as the area of pressure distribution increased.¹² This was the first study that showed a possibility to prevent ventricular fibrillation. Drewniak et al. concluded that chest protectors become more effective as their ability to distribute forces increases and, through extrapolation, a chest protector capable of distributing forces to an area of 2,500 mm² or greater could potentially eliminate the risk of CC.¹²

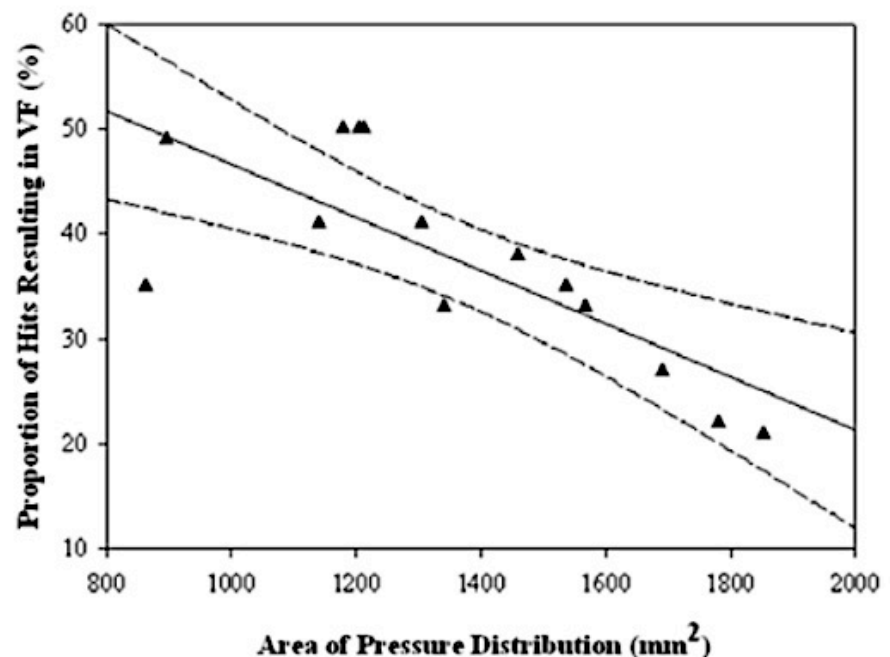


Figure 17. Linear regression model demonstrating that an increase in the area of pressure distribution significantly correlates with a decreased occurrence of ventricular fibrillation in swine models.¹⁷

Using data from various studies, including Kumar et al., Mark S. Link, M.D. teamed up with the CEO of Unequal technologies, a protective sportswear company. Together they set out to create a chest protector effective in preventing CC. Approximately five years later, the composite they developed was sent to Tufts Medical Center for testing.¹⁰ In 2015, Kumar et al. tested the chest protector prototypes created by Link and Unequal technologies and their effectiveness in preventing sudden cardiac death by chest wall impact (commotio cordis).¹⁶ Kumar et al. tested 12 different chest protectors, using impacts on swine with lacrosse balls traveling at 40 MPH. The prototypes tested were all created with the same materials, including closed-cell high density foam, closed-cell lighter density soft foam, and open cell memory foam, with a semi-rigid propylene polymer.¹⁶ The only difference between the 12 chest protectors was

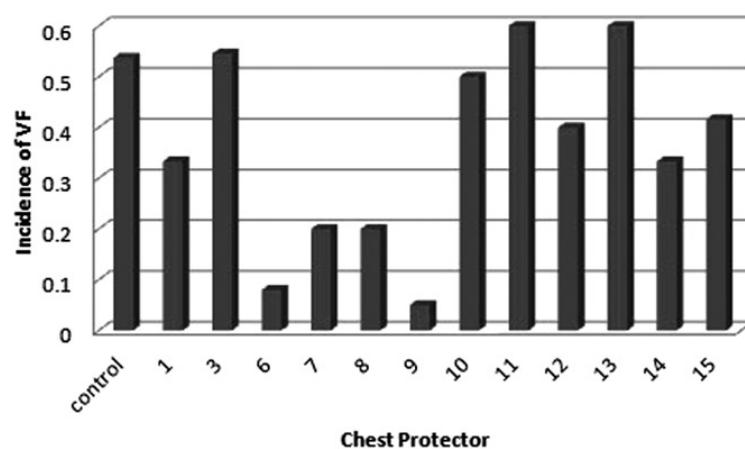


Figure 18. Incidence of ventricular fibrillation with the 12 chest protectors tested and the control. Numbers 6, 8, and 9 showed a statistically significant decrease.¹⁸

the varying thickness of the materials. It was found that only three out of the 12 tested showed significant reduction in the risk of ventricular fibrillation, and there was a linear correlation between the thickness of the chest protector and its protection.¹⁶ One chest protector tested decreased ventricular

fibrillation to only five percent of impacts, compared to 54% of impacts in the control (no chest protection).¹⁶

Following testing, Kumar et al. at Tufts Medical Center concluded that “It is reasonable to expect that chest protector designs incorporating these novel materials (Unequal HART) will

be effective in the prevention of Commotio Cordis on the playing field.”¹⁷ This was a breakthrough for CC prevention because previously, all chest protectors commercially available in 2002 and 2012 did not reduce ventricular fibrillation incidence in testing.¹⁶ In the span of ten years, there were no advances in the prevention of CC with more effective chest protectors. Ample research was being done on the topic, but no results differed from previous studies and data. The chest protector created by Link and Unequal Technologies, and tested by the Tufts Medical Center was made commercially available in 2017.



Figure 20. Non-sport specific chest protectors sold by Unequal Technologies. HART® Shirt (above),²¹ and HART® Pad (below).²²



The National Operating Committee on Standards for Athletic Equipment (NOCSAE) is a non-profit organization that sets thorough standards and certifications for equipment in order to reduce athletic injuries and death. The CEO of NOCSAE released a statement in regards to the new research and discoveries made, saying, “This is an unprecedented breakthrough in sports protection. Scientists have pinpointed the exact cause of commotio cordis, including the critical moment of occurrence in the cardiac cycle and the required threshold to prevent this injury. These findings have allowed us to create a chest protector standard that will significantly reduce, if not



Figure 19. Sport specific chest protectors sold by Unequal Technologies. HART® Catcher Chest Protector for baseball and softball catchers (above),¹⁹ and HART® Goalie Chest Protector for lacrosse goalies (below).²⁰



eliminate, the risk of commotio cordis.”¹⁸ In January 2016, NOCSAE introduced the first standard for baseball/softball and lacrosse chest protectors for commotio cordis.¹⁹ This standard, made effective January 31st, 2018, is meant to provide “reliable and repeatable measurements for the evaluation of various types of chest protectors.”¹⁹ Since January 31st 2018, there have been two updates to the standard, with the latest revision in February 2018, effective February 2019.¹⁹

In accordance with the February 2019 standard, the chest protector must be tested on a NOCSAE Thoracic Surrogate, with underlying load cells to measure the force at impact, using projectiles moving at 30 and 50 MPH.¹⁹ The projectile used must match the intended sport of the chest protector, either a lacrosse ball or a baseball. If the peak forces measured during testing are less than the maximum peak force allowed by NOCSAE, the chest protector meets NOCSAE standards. All NOCSAE approved chest protectors must have the NOCSAE sport specific seal on the exterior of the chest protector, and must be legible.¹⁹

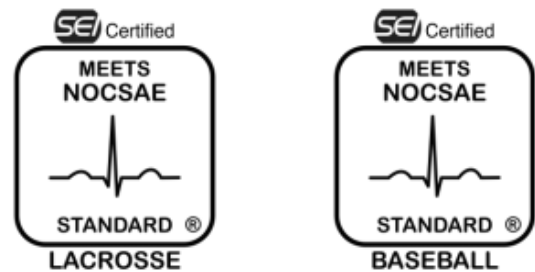


Figure 21. Sports specific NOCSAE chest protector seals for lacrosse and baseball.²³

Prevention of CC through protection and safety equipment should not stop at chest protection; the projectile can be modified as well to protect the athlete. Age appropriate “safety baseballs,” or Reduced Injury Factor (RIF), have a softer core than standard baseballs.^{3, 6, 7} There are three different levels of RIF baseballs, RIF 1, RIF 5, and RIF 10, each corresponding to how soft the core of the ball is.⁹ Each ball is designed for a specific age and skill group of athletes. RIF 1 is the softest, designed for children with the lowest skill level, RIF 5 is medium-soft, designed for youths with moderate skill level, and RIF 10 is the hardest safety ball, designed for older athletes with higher skill level.^{9, 20}

Link et al. performed a study to determine if risk of CC decreased with the use of safety baseballs.⁹ 40 animal models were used, each receiving multiple blows at 40MPH with one of

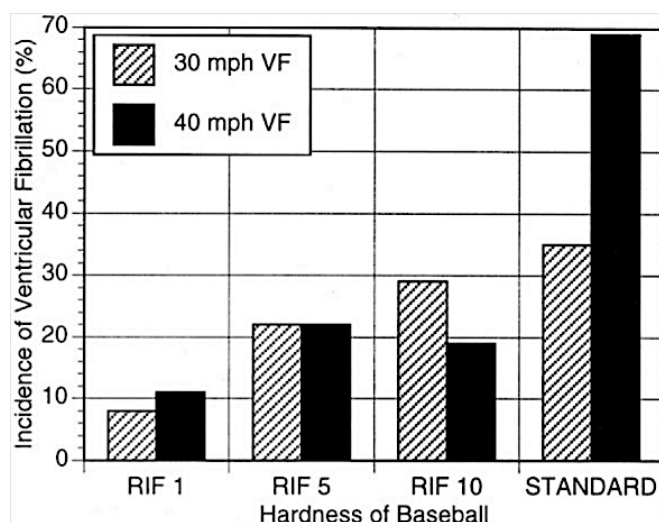


Figure 22. Incidence of ventricular fibrillation with baseballs of differing hardness at impacts of 30 and 40 MPH.²⁴

three possible safety baseballs, and a standard baseball.⁹ A linear correlation with the hardness of the baseball and the risk of ventricular fibrillation was found.

Ventricular fibrillation only occurred in 11% of impacts with RIF 1 balls, 22% with RIF 5 balls, and 19% with RIF 10 balls, compared to 69% with a standard baseball.⁹

Link et al. concluded that safety baseballs reduced, but did not eliminate, the risk of

CC, and that a more widespread use of these safety balls would help decrease the risk of sudden cardiac death for young athletes.⁹

The Consumer Product Safety Commissioner recommends the use of safety baseballs as a way to decrease the incidence of youth baseball injuries.^{3,7} NOCSAE has set standards for newly manufactured baseballs since before June 2005.²⁰ It has been updated and revised multiple times since 2005, with the latest revision in February 2018.²⁰ These standards are meant to specify the weight, compression deflection load (CDL),

circumference, and coefficient of restitution for new baseballs manufactured.²⁰ Every category of safety baseball has the same specifications for weight requirement and circumference, however the CDL differs dramatically for each. RIF 1 CDL must not exceed 45 lbs., RIF 5 CDL



Figure 23. NOCSAE seal for baseballs.²⁵

must be within 75 to 150 lbs., and RIF 10 CDL must be within 200 to 350 lbs.²⁰ The coefficient of restitution is modified for RIF 1, within 0.45 to 0.555, while RIF 5 and RIF 10 must both be within 0.5 to 0.555.²⁰ All NOCSAE certified baseballs must have the NOCSAE seal legible on the baseball's external packaging.²⁰

Despite the vast evidence showing the effectiveness of safety baseballs, there is still some resistance to their use. Many people are reluctant to use safety baseballs in organized youth baseball because they believe they alter the fundamentals of baseball.² These people claim that the bounces of these balls are unpredictable and exaggerated, and have lower velocities than standard baseballs when struck with a bat.^{6,9} However, these effects are most frequently seen with the softest, RIF 1 balls, which are most commonly designated for T-ball with athletes ages seven and younger. In fact, the RIF 5 and RIF 10 balls largely maintain the qualities of standard baseballs and should therefore be used without hesitation or reluctance, for the safety of the athletes.^{6,9}

Other suggestions that come up frequently in research for preventing CC in athletes revolve around education. Teaching athletes how to properly protect themselves should be the responsibility of parents and coaches. Athletes, especially young athletes, should be instructed to properly turn away from wild balls while batting or holding up their glove or mitt to protect their chest while fielding.⁷⁻⁹ This will help shield the athlete's chest from the ball, possibly eliminating the risk of CC because the projectile will not impact the chest wall. Palacio et al. has even suggested a change in the rules, such as eliminating chest blocking of shots in lacrosse.⁷ This could potentially help reduce the risk of CC in lacrosse defenders by removing the entire situation of players voluntarily getting struck in the chest by a ball in order to block a shot.

Conclusion

Commotio cordis is a rare yet potentially fatal event. Young athletes are more susceptible to CC due to their pliable, underdeveloped chest wall. It is the second most common cause of death in young athletes. After a projectile strikes an athlete over the precordial area and ventricular fibrillation occurs, the individual has minutes before their chance of mortality is greater than their chance of survival. Survival of CC relies on immediate recognition, and early CPR and defibrillation with an AED. Increased awareness and education of CC, as well as CPR and AED training is imperative. As these survival factors continue to improve, so too will survival rates of CC. Victims of CC who survive do not have any long-term effects from the incident, unless brain or organ damage occurred from delayed resuscitation and defibrillation. Survivors can return to play upon passing a thorough cardiac evaluation to rule out any structural heart disease. All return to play decisions should be considered case by case. Rates of CC reoccurrence are extremely low due to the nature of the event, but it has been reported. Prevention may be enhanced through the use of safety baseballs and effective chest protectors. While the technology used in chest protectors has advanced greatly in the last three years, until a chest protector can be developed that completely eliminates the risk of CC, it can still occur. Proper use of safety equipment is imperative to lowering the incidence rate of CC.

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